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## The art of medicine Stereotype threat

Among the disturbing statistics to have emerged during the COVID-19 pandemic has been the disproportionate impact in terms of death and severe illness on ethnic minorities in the UK and the USA. On April 7, 2020, it was reported that in the US city of Chicago, where the black population is roughly 30%, nearly 70% of COVID-19 deaths were in this demographic. A report released by the UK Intensive Care National Audit and Research Centre on April 17, 2020, showed that 34% of patients in the UK receiving advanced respiratory support were non-white, despite the non-white population nationally being about 14%. That day, the UK Government announced a review into the impact of COVID-19 on black, Asian, and minority ethnic communities. On May 7, 2020, a provisional analysis by the UK Office for National Statistics of NHS England data suggested that, after adjustment for age, some sociodemographic characteristics, and measures of self-reported health and disability, black people were almost twice as likely as white people to have a COVID-19-related death.

We don't yet have the full picture of this pandemic. Even so, teasing apart local demographics and infection patterns seems likely to explain at least a portion of the disparities seen to date. 44% of National Health Service medical staff, on the front line of virus exposure, are non-white. In London, one of the worst affected regions of the UK, white British people are in a minority according to the 2011 Census. Socioeconomic factors may well account for some of the disparities, as might racism in health care. However, other far more speculative explanations have also been offered: some medical researchers have raised the possibility that innate genetic differences between racial groups cause the virus to hit some harder than others.

Such speculation runs the risk of forgetting that the demographic categories we recognise socially do not in fact have very much biological meaning and betrays a wider problem in medicine when it comes to race. It has become routine in medical research and clinical practice to categorise people by race and ethnicity. While this is no doubt important in identifying demographic groups who might be disadvantaged by unequal treatment and to spot any environmental or social patterns affecting disease prevalence, these categories are also sometimes used to guide research, diagnosis, and treatment in ways that are not necessarily useful. At worst, they may be reinforcing damaging myths about biological differences between groups.

In making the case for the possibility of innate biological health differences between groups during the COVID-19 crisis, at least one researcher has pointed to the already-recognised increased risk of hypertension among black people of Afro-Caribbean descent in the UK and the USA. Hypertension is an example of a health condition that has been unambiguously racialised. It is so widely accepted as such that the UK National Institute for Health and Care Excellence guidelines recommend that black patients younger than 55 years with hypertension be given calcium-channel blockers instead of angiotensin-converting enzyme inhibitors (ACE) inhibitors, which are given to non-black patients under 55 years.

What justifies this distinction in treatment on the basis of race? When epidemiologist Jay Kaufman, at McGill University in Canada, and cardiologist and global expert on hypertension Richard Cooper, at Loyola University Chicago in the USA, analysed studies that claimed to see racial differences in responses to ACE inhibitors, they did not find evidence that black or white patients were significantly advantaged by different prescriptions. Their conclusion about the benefit of assigning ACE inhibitors according to race was that from "the point of view of any individual patient, this is not meaningfully better than being assigned by the flip of a coin". Kaufman and Cooper's research affirmed what has long been known by population geneticists. Humans are a highly homogeneous species, even more so than our closest evolutionary cousin, the chimpanzee. By far the greatest source of human genetic variation is not group differences, but individual differences. This is perhaps why, for all the effort that has been poured into research to prove the long-held hypothesis that racial differences seen in hypertension have a genetic basis, scientists have not found anything consistent in our DNA to support it.

More pertinently, when we talk about race we are talking about groups that are socially defined. In the USA,

### Further reading

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for instance, someone may have just one grandparent of African ancestry but be categorised as black based on appearance. It makes little sense to conduct research around the assumption that a socially defined group could exhibit a genetic difference from another socially defined group when the groups are not biologically defined to begin with. To do so defies logic. It similarly defies logic to assume that all non-white people in the UK, with their diverse geographical ancestries, are so genetically different from white people that they will as a group be more innately affected by COVID-19.

We need only look to history to understand how race was constructed and how little grounding it ever had in biology. One reason counter-assumptions persist is that racial categories are such looming presences in our social and cultural lives that we can't imagine they don't have a firmer biological basis. This is not to say that group-level differences do not exist—for example, in certain genetic conditions that run in families—and indeed, such research should not be dismissed if it can yield useful insights. But it must also be remembered that where such variations are seen, they are fuzzy and marginal, and cannot be a reliable guide to the treatment of any one patient. Very often, environmental and social factors are at play rather than biological ones. In the case of hypertension, one dominant risk factor is diet, particularly salt consumption. Alongside other known social risk factors such as physical inactivity and obesity, research looking at hypertension in black Americans suggests that there might also be an association with stress, possibly including the stress associated with racial discrimination.

Yet, time and again, I have seen health researchers erroneously invoke social categories as though they are biological ones. At a conference on diversity in clinical trials held in London, UK, I saw an employee from a major pharmaceutical company repeatedly refer to “Latino” as an “ancestral” group. Anyone familiar with the history of the Americas will know that this socially defined group of people with cultural ties to Latin America comprises individuals of hugely diverse ancestries. Even at a stretch it can't be considered a genetically similar group for the purposes of scientific research. Incidents like this have made me wonder whether some well intentioned but misguided medical researchers are keeping race science alive.

The temptation to group people by perceived common traits is known in any other sphere of life as stereotyping. I would argue that in medicine there is also a dangerous habit of racial stereotyping. This tendency to treat people in the same social group as similar, to enter into biological essentialism, too easily glosses over the complexities and breadth of individual differences.

One possible reason that race keeps being reintroduced into health research is that one of its blue-sky goals is to have personalised medicine so precise that every person's biological profile is perfectly understood, avoiding adverse

drug reactions and unnecessary deaths. In the greyer real world in which we don't have all the necessary data to do this, some doctors and researchers instead turn to social categories as proxies. They work on the assumption that certain groups share certain health traits on average, allowing them to roughly gauge the medical requirements of any patient belonging to that group. It feels like a useful step on the road to personalised medicine, some might argue. In my view, it is a fudge. Our social categories have enormous power in society, in dictating how we live and how we are treated by others, but this doesn't mean they have the same significance underneath our skins.

When gaps are seen between groups, we must therefore be careful about diagnosing the causes until we have clear evidence. Casual speculation helps nobody. The UK Confidential Enquiry into Maternal Deaths found in 2018 that black women were five times more likely than white women to die during pregnancy and childbirth. Similar mortality rates have been seen in the USA, not only among black women but also other minorities. Some have wondered whether this might have something to do with black women's bodies being in some way more vulnerable and less able to withstand pregnancy. It has fallen on health professionals working with minority women, such as the brilliant Florida-based midwife Jennie Joseph, to show through on-the-ground work with pregnant women that, simply by improving standards of care, mortality rates can be drastically improved. It is not groups of people that are different, Joseph's work proves, but how those groups are treated. It is racism that kills, not race.

Scientists are finally investigating the damaging effects of conscious and unconscious bias on patients, revealing disturbing truths about racism, sexism, and other forms of prejudice in medicine. A review of gender differences in the treatment of chronic pain in 2018 showed that women tend to be taken less seriously than men, and are more easily dismissed as over-sensitive or hysterical. A 2020 study uncovered racial disparities in dementia care in the UK, with Asian dementia patients less likely to be prescribed anti-dementia drugs than white patients.

These are not easy facts to confront for doctors and medical researchers who believe they are doing their best for patients. But the persistent habit of essentialising large groups of people in medicine, I would argue, needs to be interrogated. Medical research is not helped by maintaining myths. If race is to be used as a research variable or diagnostic tool, the reasons why need to be clearly articulated and justified, to avoid a reliance on stereotypes rather than on facts.

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